

Endometriosis & Polycystic Ovarian disease

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Objective

By the end of this lecture the students must be able to :

- Identify the definition, pathogenesis, pathological finding & clinical finding of endometriosis.
- Describe polycystic ovarian disease

Endometriosis

- Defined by the presence of endometrial glands and stroma in a location **outside** the endometrium.
- Usually it is **multifocal involving pelvic structures** (ovaries, pouch of Douglas, uterine ligaments, tubes, and rectovaginal septum).
- Less frequently involves peritoneal cavity or periumbilical tissues.
- Rarely, involve lymph nodes, lungs, heart, skeletal muscle, or bone.

Occurrence

- Typical patient with endometriosis is in her **reproductive** years (10%), and **sub-fertile**.
- Pathogenesis of endometriosis is not completely understood and believed to be a combination of factors.

Theories of pathogenesis

- **Regurgitation theory**

(Retrograde menstruation)

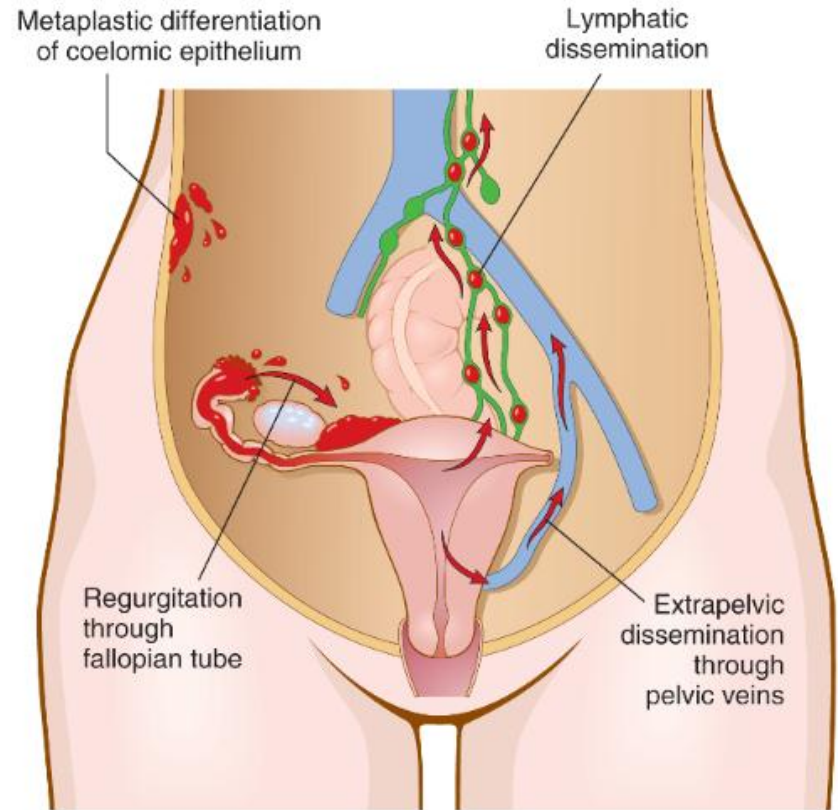
Endometrial fragments transported through fallopian tubes at time of menstruation and implant at intra-abdominal sites

- **Metaplastic theory**

Metaplastic transformation of pelvic peritoneum

- **Vascular or lymphatic dissemination theory**

Substances released/shed from endometrium induce formation of endometriosis

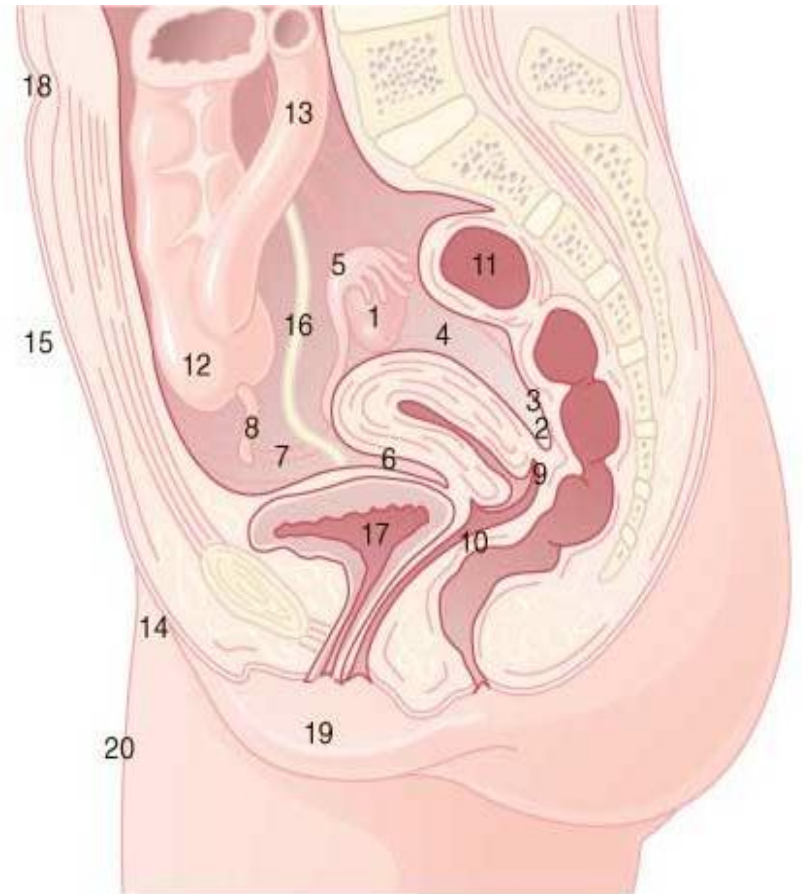


pathogenesis of endometriosis

- Recently found unlike normal endometrium any *misplaced* endometriotic tissue **abnormally exhibit increased levels of inflammatory mediators** (prostaglandin E2, and increased estrogen production) that enhance the survival and **persistence of the endometriotic tissue** within a foreign location

Site of occurrence

- Ovary (most common)
- Cul-de-sac
- Uterosacral ligaments
- Broad ligament
- Fallopian tubes
- Round ligaments
- Vagina
- Rectosigmoid and bowel, appendix
- Urinary bladder and ureters



Endometriosis

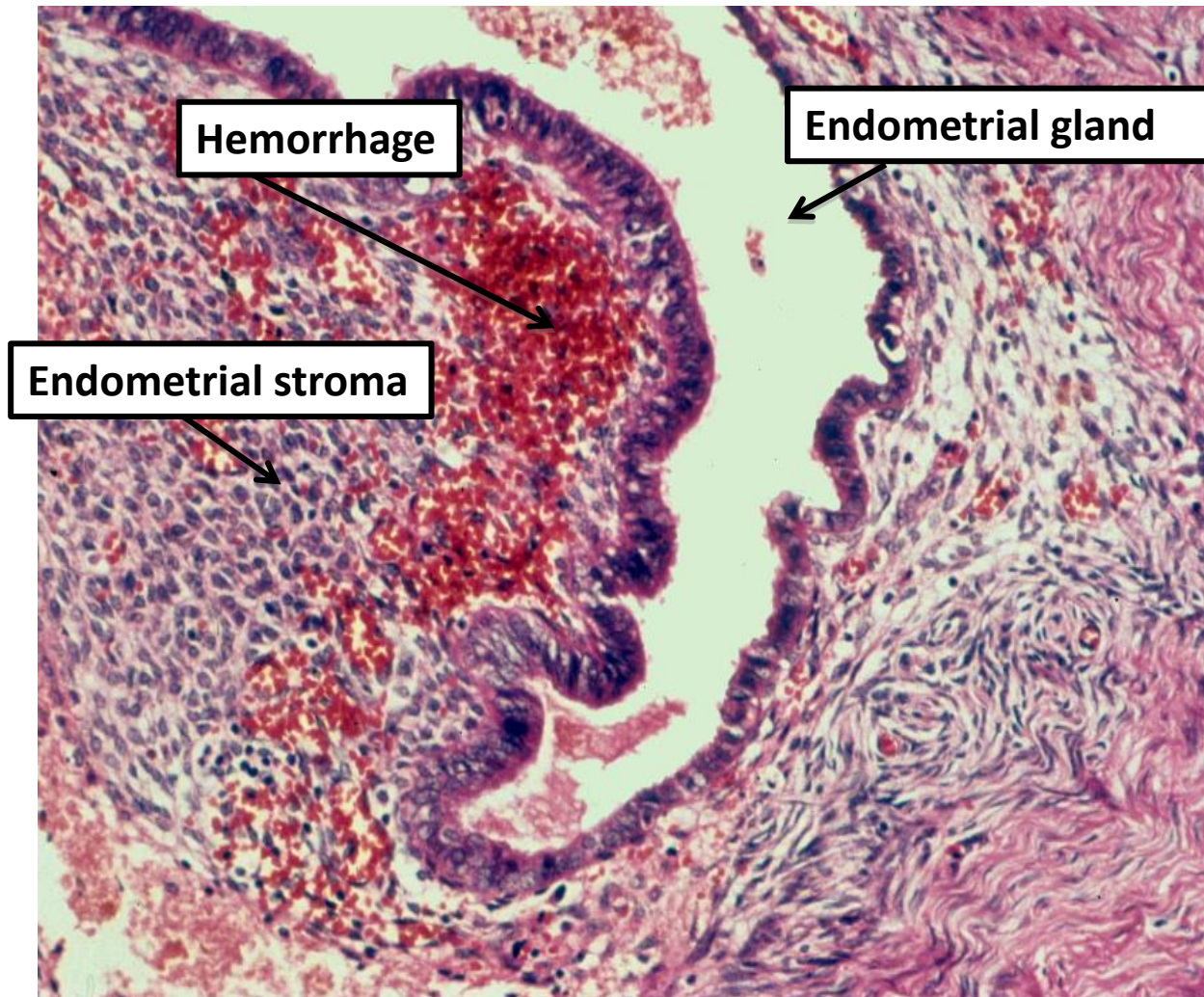
- The **functioning endometrium** undergoes **cyclic bleeding**.
- **End by fibrosis, pelvic adhesion**, sealing of the tubal fimbriated ends, distortion of the oviducts and ovaries seepage (d.t. blood organization) .

Pathology

Grossly : Multiple
endometrial cysts
“**chocolate cysts**”
red brown
nodules of the
ovary



The histologic diagnosis



At all sites, depends on finding two of the following three features within the lesions:

- **Endometrial glands,**
- **Endometrial stroma,**
- **Hemosiderin pigment.**

Clinical Features

In endometriosis with cyclic bleeding.

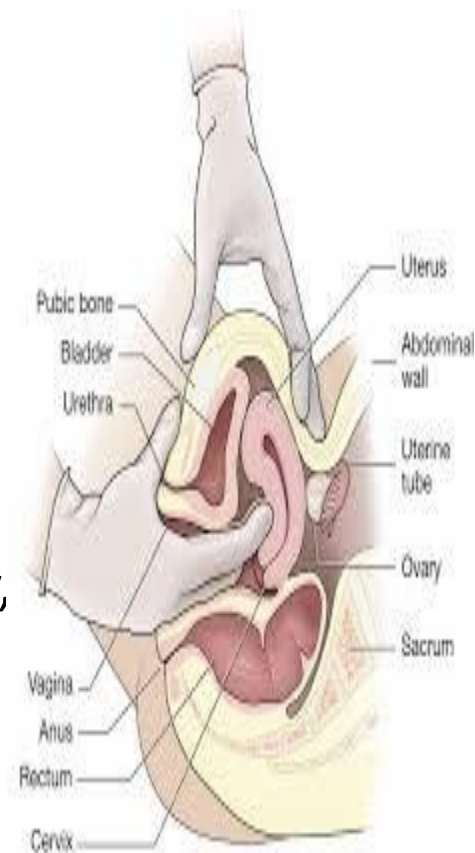
Characteristic triad of symptoms is associated
(**dysmenorrhea**, **dyspareunia**, & **dyschezia**).

Depending on the distribution of the endometriotic lesions :

- **Severe dysmenorrhea and pelvic pain** (intra-pelvic bleeding and peri-uterine adhesions).
- **Dyschezia, Pain on defecation** (Rectal wall involvement)
- **Dyspareunia**, painful intercourse (uterine involvement)
- **Dysuria** (bladder involvement).
- **Sterility** (extensive scarring of the oviducts and ovaries).
- **Staging of endometriosis is not clearly associated with frequency and severity of pain symptoms.**
- There is a **risk of recurrence** of endometriosis throughout a woman's life.

Diagnosis Should be suspected in :

- An **afebrile** patient with the **characteristic triad** of pelvic pain, a firm, fixed, tender adnexal mass.
- **Tender nodularity** in the cul-de-sac and uterosacral ligaments in uterine bimanual examination). The characteristic sharp, firm, exquisitely **tender "barb"** (from barbed wire) felt in the uterosacral ligament is diagnostic finding in severe cases.
- **Ultrasound** – adnexal mass of complex echogenicity, internal echoes consistent with blood.
- **Definitive diagnosis** is made by the characteristic **gross and histologic findings** obtained at laparoscopy or laparotomy.



Treatment

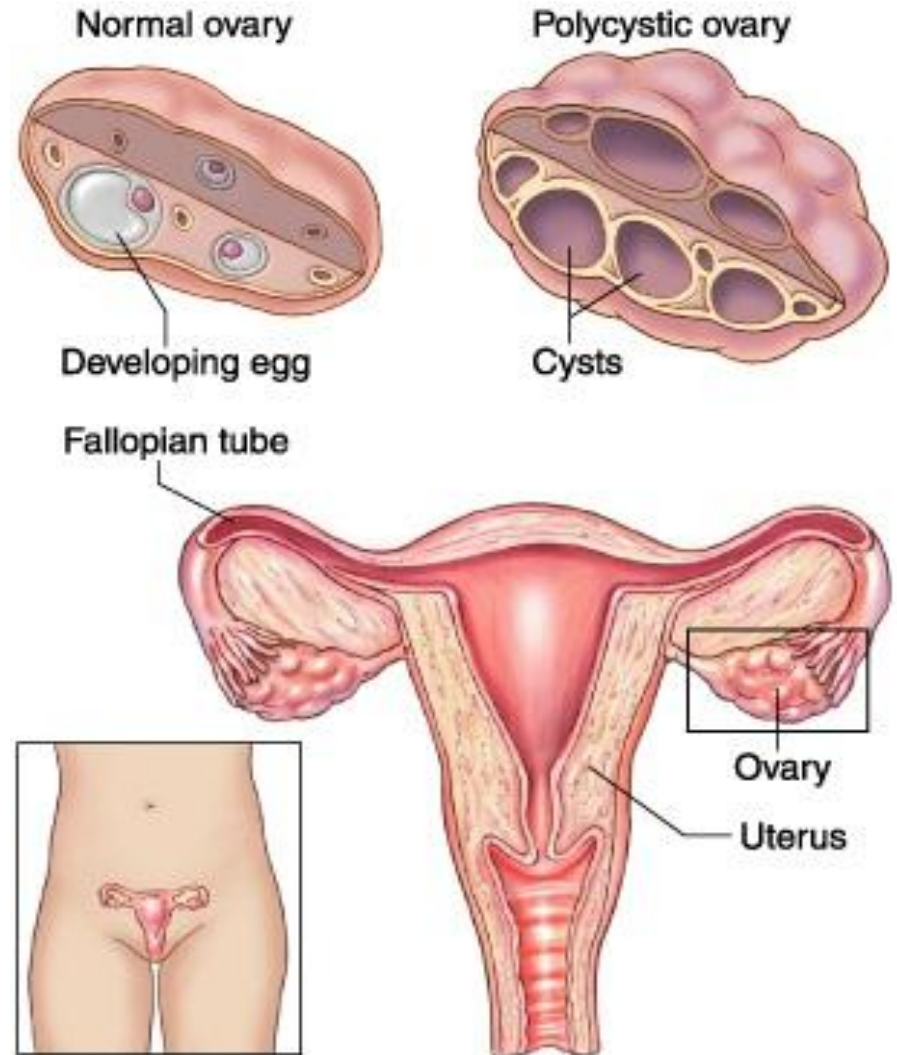
- Treatment is **indicated for** endometriosis associated **pelvic pain**, dysmenorrhea, dyspareunia, abnormal bleeding, ovarian cysts, and infertility due to gross distortion of tubal and ovarian anatomy.
- Appropriate treatment **varies** widely and should take into consideration severity of symptoms, extent of disease, desire for future fertility & patient age.
- In all women, **minimization of menstrual flow and suppression of ovarian cycling** can reduce the risk for endometriosis.

ADENOMYOSIS

- **Growth** of the basal layer, **stratum basalis**, of the endometrium **down into the myometrium**.
- Forming nests of endometrial stroma, glands, or both that induce reactive endometrium hypertrophy & **uterine enlargement**, .
- Unlike endometriosis there is **no cyclic bleeding**.
- Menorrhagia, pelvic pain before the onset of menstruation.

Polycystic ovarian disease

- A disorder in which **multiple cystic follicles** in the ovaries produce **excess androgens** and **estrogens**.



Polycystic ovarian disease

- It usually comes to attention after menarche in **teenage girls** or young adults who present with **oligomenorrhea**, **hirsutism**, **infertility**, and sometimes **obesity**.
- The origins of these changes are poorly understood, but it is proposed that the ovaries elaborate excess androgens, which are converted to estrogenic hormones in peripheral fatty depots, which inhibit the secretion of follicle-stimulating hormone.
- http://www.youtube.com/watch?v=FsNKyKS7M_s

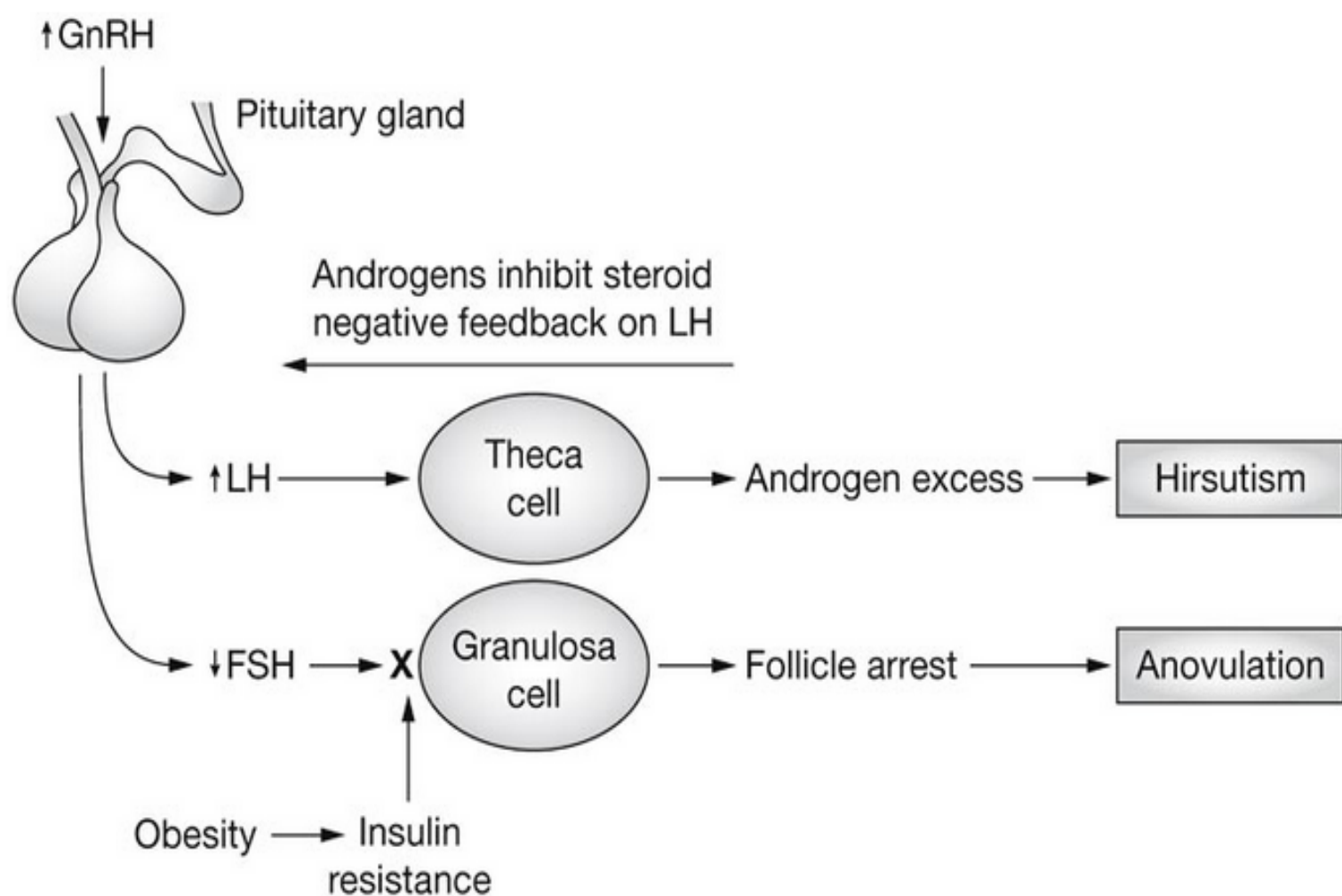
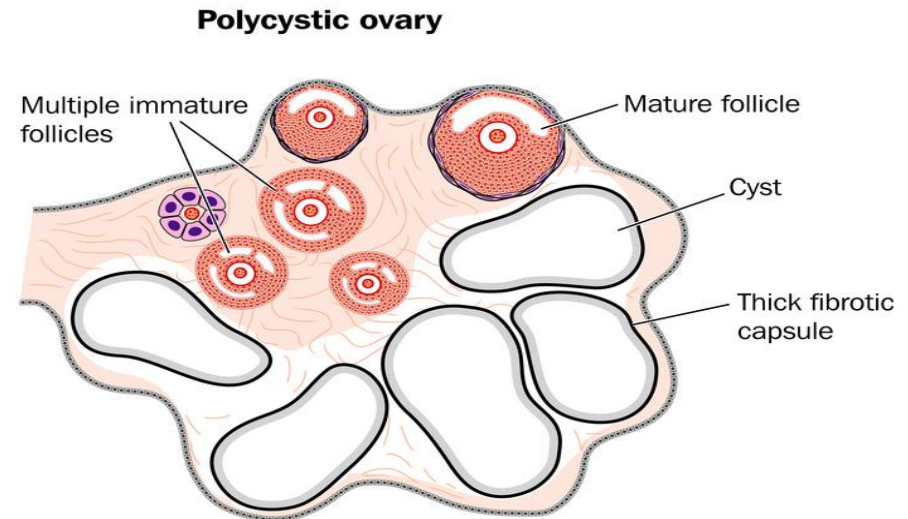
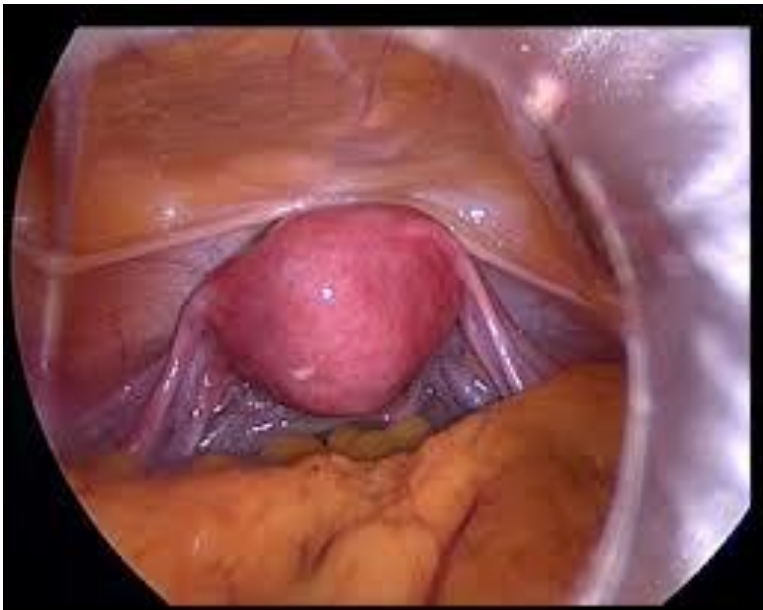


Figure 1. Pathophysiology of polycystic ovary syndrome (PCOS).

Diagnostic features of PCOS are hirsutism, anovulation, and polycystic ovaries, which show arrested follicular maturation; obesity and insulin resistance are frequently associated conditions. The major biochemical feature of PCOS is androgen excess, which causes hirsutism. Androgens also appear to inhibit the negative-feedback effects of estrogens and progesterone on pulsatile LH release. Women with PCOS have increased pulsatile GnRH release, which results in higher levels of LH and lower levels of FSH in most individuals. Higher LH (and insulin) levels seem to cause increased androgen production by follicular theca cells whereas lower FSH levels lead to anovulation. Obesity and insulin resistance decrease levels of sex-hormone-binding globulin and thereby increase testosterone bioactivity. If follicular granulosa cells are insulin resistant, it might affect their responses to FSH; otherwise, granulosa cells appear to be very capable of releasing estrogen in response to FSH, perhaps as a result of the actions of androgens and insulin. Abbreviations: FSH, follicle-stimulating hormone; GnRH, gonadotropin-releasing hormone; LH, luteinizing hormone.

Polycystic ovarian disease

- **Macroscopically** the ovaries are usually twice the normal size, gray-white with a smooth outer cortex, and studded with subcortical cysts 0.5 to 1.5 cm in diameter.
- **Histologic examination** reveals a thickened, fibrotic ovarian capsule overlying innumerable cystic follicles lined by granulosa cells. There is a conspicuous absence of corpora lutea in the ovary.



REFERENCE BOOK

- Robbins Basic Pathology : PAGE (689-691 & 695- 696)